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|---|---|---|
| NEWS 1 | | Web Page URLs for STN Seminar Schedule - N. America |
| NEWS 2 | Apr 08 | "Ask CAS" for self-help around the clock |
| NEWS 3 | Jun 03 | New e-mail delivery for search results now available |
| NEWS 4 | Aug 08 | PHARMAMarketLetter(PHARMAML) - new on STN |
| NEWS 5 | Aug 19 | Aquatic Toxicity Information Retrieval (AQUIRE) now available on STN |
| NEWS 6 | Aug 26 | Sequence searching in REGISTRY enhanced |
| NEWS 7 | Sep 03 | JAPIO has been reloaded and enhanced |
| NEWS 8 | Sep 16 | Experimental properties added to the REGISTRY file |
| NEWS 9 | Sep 16 | CA Section Thesaurus available in CAPLUS and CA |
| NEWS 10 | Oct 01 | CASREACT Enriched with Reactions from 1907 to 1985 |
| NEWS 11 | Oct 24 | BEILSTEIN adds new search fields |
| NEWS 12 | Oct 24 | Nutraceuticals International (NUTRACEUT) now available on STN |
| NEWS 13 | Nov 18 | DKILIT has been renamed APOLLIT |
| NEWS 14 | Nov 25 | More calculated properties added to REGISTRY |
| NEWS 15 | Dec 04 | CSA files on STN |
| NEWS 16 | Dec 17 | PCTFULL now covers WP/PCT Applications from 1978 to date |
| NEWS 17 | Dec 17 | TOXCENTER enhanced with additional content |
| NEWS 18 | Dec 17 | Adis Clinical Trials Insight now available on STN |
| NEWS 19 | Jan 29 | Simultaneous left and right truncation added to COMPENDEX, ENERGY, INSPEC |
| NEWS 20 | Feb 13 | CANCERLIT is no longer being updated |
| NEWS 21 | Feb 24 | METADEX enhancements |
| NEWS 22 | Feb 24 | PCTGEN now available on STN |
| NEWS 23 | Feb 24 | TEMA now available on STN |
| NEWS 24 | Feb 26 | NTIS now allows simultaneous left and right truncation |
| NEWS 25 | Feb 26 | PCTFULL now contains images |
| NEWS 26 | Mar 04 | SDI PACKAGE for monthly delivery of multifile SDI results |
| NEWS 27 | Mar 20 | EVENTLINE will be removed from STN |
| NEWS 28 | Mar 24 | PATDPAFULL now available on STN |
| NEWS 29 | Mar 24 | Additional information for trade-named substances without structures available in REGISTRY |
| NEWS 30 | Apr 11 | Display formats in DGENE enhanced |
| NEWS 31 | Apr 14 | MEDLINE Reload |
| NEWS 32 | Apr 17 | Polymer searching in REGISTRY enhanced |
| NEWS 33 | Apr 21 | Indexing from 1947 to 1956 being added to records in CA/CAPLUS |
| NEWS 34 | Apr 21 | New current-awareness alert (SDI) frequency in WPIDS/WPINDEX/WPIX |
| NEWS 35 | Apr 28 | RDISCLOSURE now available on STN |
| NEWS 36 | May 05 | Pharmacokinetic information and systematic chemical names added to PHAR |
| NEWS 37 | May 15 | MEDLINE file segment of TOXCENTER reloaded |
| NEWS 38 | May 15 | Supporter information for ENCOMPPAT and ENCOMPLIT updated |
| NEWS 39 | May 16 | CHEMREACT will be removed from STN |
| NEWS 40 | May 19 | Simultaneous left and right truncation added to WSCA |
| NEWS 41 | May 19 | RAPRA enhanced with new search field, simultaneous left and right truncation |

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 NEWS EXPRESS  April 4 CURRENT WINDOWS VERSION IS V6.01a, CURRENT
               MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),
               AND CURRENT DISCOVER FILE IS DATED 01 APRIL 2003
 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS INTER    General Internet Information
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 NEWS PHONE    Direct Dial and Telecommunication Network Access to STN
 NEWS WWW      CAS World Wide Web Site (general information)

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FILE 'HOME' ENTERED AT 14:36:47 ON 19 MAY 2003

=> file medline caplus biotechno scisearch biosis
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

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FILE 'BIOSIS' ENTERED AT 14:37:23 ON 19 MAY 2003
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=> s tgf?
L1      108705 TGF?

=> s l1 and ecm
L2        1828 L1 AND ECM

=> sl1 and extracellular matrix
SL1 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s l1 and extracellular matrix
L3       10238 L1 AND EXTRACELLULAR MATRIX

=> s l2 or l3
L4       10325 L2 OR L3

=> s l4 and ACE inhibitor#
L5          76 L4 AND ACE INHIBITOR#
```

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6 40 DUP REM L5 (36 DUPLICATES REMOVED)

=> d ti 1-40

L6 ANSWER 1 OF 40 MEDLINE DUPLICATE 1
TI Association of transforming growth factor-beta (**TGF**-beta) T869C (Leu 10Pro) gene polymorphisms with type 2 diabetic nephropathy in Chinese.

L6 ANSWER 2 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Angiotensin-converting enzyme inhibitor attenuates pancreatic inflammation and fibrosis in male Wistar Bonn/Kobori rats

L6 ANSWER 3 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Preemptive ramipril therapy delays renal failure and reduces renal fibrosis in COL4A3-knockout mice with Alport syndrome

L6 ANSWER 4 OF 40 CAPLUS COPYRIGHT 2003 ACS
TI ACE inhibition increases expression of the ETB receptor in kidneys of mice with unilateral obstruction

L6 ANSWER 5 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Add-on angiotensin II receptor blockade lowers urinary transforming growth factor-beta levels

L6 ANSWER 6 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Decreased matrix degradation in diabetic nephropathy: effects of ACE inhibition on the expression and activities of matrix metalloproteinases

L6 ANSWER 7 OF 40 MEDLINE DUPLICATE 2
TI Angiotensin II and renal fibrosis.

L6 ANSWER 8 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Angiotensin converting enzyme inhibitor suppresses glomerular transforming growth factor beta receptor expression in experimental diabetes in rats

L6 ANSWER 9 OF 40 MEDLINE DUPLICATE 3
TI Urinary tract obstruction.

L6 ANSWER 10 OF 40 MEDLINE DUPLICATE 4
TI **ACE inhibitors** attenuate expression of renal transforming growth factor-beta1 in humans.

L6 ANSWER 11 OF 40 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI Plasma levels of transforming growth factor (**TGF**)-beta1 as a predictive marker in diabetic nephropathy.

L6 ANSWER 12 OF 40 MEDLINE DUPLICATE 5
TI Potential contribution of a novel antifibrotic factor, hepatocyte growth factor, to prevention of myocardial fibrosis by angiotensin II blockade in cardiomyopathic hamsters.

L6 ANSWER 13 OF 40 MEDLINE DUPLICATE 6
TI Age-related progressive renal fibrosis in rats and its prevention with **ACE inhibitors** and taurine.

L6 ANSWER 14 OF 40 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 7
TI Age-related progressive renal fibrosis in rats and its prevention with **ACE inhibitors** and taurine

L6 ANSWER 15 OF 40 MEDLINE
TI Role of angiotensin II in diabetic nephropathy.

L6 ANSWER 16 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Rationales for treating IgA nephropathies

L6 ANSWER 17 OF 40 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 8
TI Effect of Long-term ACE Inhibition on Myocardial Tissue in Hypertensive Stroke-prone Rats

L6 ANSWER 18 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Blocking angiotensin II ameliorates proteinuria and glomerular lesions in progressive mesangioproliferative glomerulonephritis

L6 ANSWER 19 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Pathological expression of renin and angiotensin II in the renal tubule after subtotal nephrectomy - Implications for the pathogenesis of tubulointerstitial fibrosis

L6 ANSWER 20 OF 40 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI Experimental interstitial nephritis.

L6 ANSWER 21 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Ventricular remodeling and transforming growth factor-beta 1 mRNA expression after nontransmural myocardial infarction in rats: effects of angiotensin converting enzyme inhibition and angiotensin II type 1 receptor blockade

L6 ANSWER 22 OF 40 CAPLUS COPYRIGHT 2003 ACS
TI Renal protective effects of blocking the intrarenal renin-angiotensin system

L6 ANSWER 23 OF 40 CAPLUS COPYRIGHT 2003 ACS
TI Angiotensin converting enzyme inhibition reduces the expression of transforming growth factor-.beta.1 and type IV collagen in diabetic vasculopathy

L6 ANSWER 24 OF 40 MEDLINE DUPLICATE 9
TI Targeting **TGF**-beta overexpression in renal disease: maximizing the antifibrotic action of angiotensin II blockade.

L6 ANSWER 25 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Expression of transforming growth factor-beta 1 and type IV collagen in the renal tubulointerstitium in experimental diabetes - Effects of ACE inhibition

L6 ANSWER 26 OF 40 MEDLINE DUPLICATE 10
TI Link between angiotensin II and **TGF**-beta in the kidney.

L6 ANSWER 27 OF 40 MEDLINE DUPLICATE 11
TI Attenuation of diabetes-associated mesenteric vascular hypertrophy with perindopril: morphological and molecular biological studies.

L6 ANSWER 28 OF 40 CAPLUS COPYRIGHT 2003 ACS
TI Angiotensin-converting enzyme inhibition attenuates proteinuria and renal **TGF**-.beta.1 mRNA expression in rats with chronic renal disease

L6 ANSWER 29 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Transforming growth factor beta 1 and renal injury following subtotal nephrectomy in the rat: Role of the renin-angiotensin system

L6 ANSWER 30 OF 40 MEDLINE DUPLICATE 12
TI Angiotensin-converting enzyme inhibition decreases growth factor expression in the neonatal rat kidney.

L6 ANSWER 31 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI

TI Mechanisms and prevention of chronic renal failure.

L6 ANSWER 32 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Comparative study of **ACE inhibitors** and angiotensin II receptor antagonists in interstitial scarring

L6 ANSWER 33 OF 40 MEDLINE DUPLICATE 13
TI Postnatal growth of the heart and its blood vessels.

L6 ANSWER 34 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI PASSIVE HEYMANN NEPHRITIS - EVIDENCE THAT ANGIOTENSIN-CONVERTING ENZYME-INHIBITION REDUCES PROTEINURIA AND RETARDS RENAL STRUCTURAL INJURY

L6 ANSWER 35 OF 40 MEDLINE DUPLICATE 14
TI ACE inhibition reduces proteinuria, glomerular lesions and **extracellular matrix** production in a normotensive rat model of immune complex nephritis.

L6 ANSWER 36 OF 40 MEDLINE DUPLICATE 15
TI Transforming growth factor-beta and angiotensin II: the missing link from glomerular hyperfiltration to glomerulosclerosis?.

L6 ANSWER 37 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI INCREASED GLOMERULAR CAPILLARY-PRESSURE ALTERS GLOMERULAR CYTOKINE EXPRESSION

L6 ANSWER 38 OF 40 MEDLINE DUPLICATE 16
TI Cilazapril suppresses myointimal proliferation after vascular injury: effects on growth factor induction in vascular smooth muscle cells.

L6 ANSWER 39 OF 40 CAPLUS COPYRIGHT 2003 ACS
TI The proliferative response to vascular injury is suppressed by angiotensin-converting enzyme inhibition

L6 ANSWER 40 OF 40 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.
TI The proliferative response to vascular injury is suppressed by angiotensin-converting enzyme inhibition

=> d ab 10 23 24 25 26 28 32 35 39

L6 ANSWER 10 OF 40 MEDLINE DUPLICATE 4
AB Progressive nephropathies are characterized by the enhanced accumulation of **extracellular matrix** in the kidney. Overproduction of transforming growth factor-beta (**TGF**-beta) was shown to result in pathological tissue fibrosis through the accumulation of **extracellular matrix** proteins. It has been proposed that angiotensin II stimulates **TGF**-beta production. Despite accumulating data supporting the effects of angiotensin-converting enzyme (**ACE**) **inhibitors** on the attenuation of **TGF** -beta in vitro and in rats, such studies in humans are lacking. The present study sought to determine the effects of **ACE inhibitors** on **TGF**-beta1 in patients with glomerulonephritis. Using competitive polymerase chain reaction and the sandwich enzyme-linked immunosorbent assay, **TGF**-beta1 messenger RNA (mRNA) abundance and **TGF**-beta1 protein levels were measured. Patients with immunoglobulin A nephropathy administered **ACE inhibitors** showed significantly lower renal **TGF**-beta1 gene expression than patients not administered these medications (mean ratios of **TGF**-beta1/beta-actin, 4.27 +/- 0.62 [SEM] versus 14.81 +/- 3.87; $P < 0.05$), whereas no difference was noted between patients administered **ACE inhibitors** and healthy controls (4.27 +/- 0.62 versus 2.78 +/- 0.71). **ACE inhibitor** therapy did not affect **TGF**-beta1 mRNA expression in freshly isolated

mononuclear cells. Urine and serum **TGF**-beta1 protein levels were not affected by the administration of **ACE inhibitors**. However, possibly a longer duration of treatment would decrease **TGF**-beta1 levels in urine or blood. In conclusion, we observed a significant reduction in **TGF**-beta1 expression in the kidney by **ACE inhibitors**, and this suggests that the effects of **ACE inhibitors** observed in animals can be extrapolated to patients with chronic renal disease.

L6 ANSWER 23 OF 40 CAPLUS COPYRIGHT 2003 ACS
AB The purpose of this study was to assess the role of transforming growth factor (**TGF**)-.beta.1 in the development of diabetes-assocd. mesenteric vascular hypertrophy and in the antitrophic effect of angiotensin converting enzyme inhibitors. Streptozotocin-induced diabetic and control Sprague-Dawley rats were randomly allocated to treatment with the angiotensin converting enzyme inhibitor ramipril or to no treatment and were killed 1 or 3 wk after the streptozotocin injection. Blood was collected and mesenteric vessels removed. Mesenteric vascular wt. was measured and **TGF**-.beta.1 and .alpha.1 (type IV) collagen messenger (m)RNA levels were analyzed by Northern anal. Immunohistochem. analyses for **TGF**-.beta.1 and type IV collagen were also performed. The diabetic rats had increased mesenteric vessel wt. at 3 wk but not at 1 wk and a concomitant rise in mesenteric **TGF**-.beta.1 and in .alpha.1 (type IV) collagen mRNA levels. Ramipril treatment attenuated mesenteric vessel hypertrophy and prevented the increase in **TGF**-.beta.1 and .alpha.1 (type IV) collagen mRNA levels after 3 wk of diabetes. The immunohistochem. anal. revealed that diabetes was assocd. with increased **TGF**-.beta.1 and type IV collagen protein and **extracellular matrix** accumulation in mesenteric vessels, and this increase was reduced by ramipril treatment. These results support the concept that **TGF**-.beta. is involved in the changes assocd. with diabetic vascular disease, and suggest a mechanism by which angiotensin converting enzyme inhibitors exert their antitrophic effects.

L6 ANSWER 24 OF 40 MEDLINE DUPLICATE 9
AB BACKGROUND: Overproduction of transforming growth factor-beta (**TGF**-beta) is a key mediator of **extracellular matrix** accumulation in fibrotic diseases. We hypothesized that the degree of reduction of pathological **TGF**-beta expression can be used as a novel index of the antifibrotic potential of angiotensin II (Ang II) blockade in renal disease. METHODS: One day after induction of Thy 1.1 glomerulonephritis, rats were treated with increasing doses of the Ang I converting enzyme (**ACE**) **inhibitor** enalapril and/or the Ang II receptor blocker losartan in the drinking water. Six days after disease induction the therapeutic effect on glomerular **TGF**-beta overexpression was evaluated. RESULTS: Both enalapril and losartan reduced **TGF**-beta overproduction in a dose-dependent manner, showing a moderate reduction at doses known to control blood pressure in renal forms of hypertension. A maximal reduction in **TGF**-beta expression of approximately 45% was seen for both drugs starting at 100 mg/liter enalapril and 500 mg/liter losartan, with no further reduction at doses of enalapril up to 1000 mg/liter or losartan up to 2500 mg/liter. Co-treatment with both drugs was not superior to single therapy. Consistent with our hypothesis that reduction in **TGF**-beta expression is a valid target, other disease measures, including glomerular matrix accumulation, glomerular production and mRNA expression of the matrix protein fibronectin and the protease inhibitor plasminogen-activator-inhibitor type 1 (PAI-1) closely followed **TGF**-beta expression. CONCLUSIONS: The data suggest that these therapies act through very similar pathways and that, in order to more effectively treat renal fibrosis, these drugs must be combined with other drugs that act by different mechanisms.

L6 ANSWER 25 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AB Transforming growth factor-beta (**TGF**-beta) and the renin-angiotensin system (RAS) have both been implicated in the pathogenesis of glomerulosclerosis in diabetic kidney disease, However, tubulointerstitial pathology may also be an important determinant of progressive renal dysfunction in diabetic nephropathy. In the present study, we investigated tubulointerstitial injury, **TGF**-beta 1 expression, and the effect of blocking the RAS by inhibition of ACE, We randomized 36 male SD rats to control and diabetic groups, Diabetes was induced in 24 rats by administration of streptozotocin; 12 diabetic rats were further randomized to receive the **ACE inhibitor** ramipril (3 mg/l drinking water), At 6 months, experimental diabetes was associated with tubulointerstitial damage, a 70% increase in expression of **TGF**-beta 1 ($P < 0.05$ vs, control) and a 120% increase in alpha 1 (IV) collagen gene expression ($P < 0.01$ vs. control). In situ hybridization demonstrated a diffuse increase in both **TGF**-beta 1 and alpha 1 (IV) collagen mRNA in renal tubules, In addition, intense expression of both transcripts was noted in regions of focal tubular dilatation, Administration of the **ACE inhibitor** ramipril prevented tubulointerstitial injury and the overexpression of **TGF**-beta 1 and alpha 1 (IV) collagen mRNA. Changes in gene expression were accompanied by parallel changes in immunostaining for **TGF**-beta 1 and type IV collagen, The observed beneficial effects of ramipril on the tubulointerstitium in experimental diabetes suggest that this mechanism may contribute to the therapeutic effect of **ACE inhibitors** in diabetic nephropathy.

L6 ANSWER 26 OF 40 MEDLINE DUPLICATE 10
AB Glomerulosclerosis and tubulointerstitial fibrosis are common morphological correlates of many end-stage kidneys. There is ample evidence that transforming growth factor-beta (**TGF**-beta) plays a major role in these alterations by directly stimulating synthesis of many **extracellular matrix** components and reducing collagenase production, finally leading to renal scarring. Although many factors may induce **TGF**-beta expression in the kidney, one very interesting aspect is the link between angiotensin II (ANG II) and **TGF**-beta. Originating from observations in vascular smooth muscle cells, there are now several additional studies showing that ANG II stimulates **TGF** -beta expression in the kidney. Although cell culture studies have convincingly demonstrated that the vasoactive peptide directly stimulates transcription as well as bioactivation of **TGF**-beta, the in vivo evidence is more indirect. Nevertheless, there are several pathophysiological situations including unilateral ureteral obstruction, chronic cyclosporin A nephrotoxicity, various models of hypertension, and probably diabetic nephropathy in which ANG II-mediated **TGF**-beta induction has been demonstrated to play an important role in the progression of the disease. The fascinating aspect of this relationship between ANG II and **TGF**-beta is the fact that hemodynamic changes as well as structural changes are linked together generating a unifying model of progression of chronic renal failure with ANG II as the key player. Angiotensin-converting enzyme (**ACE**) **inhibitor** and the more recently introduced AT1-receptor blocker may be potential drugs to interfere with this ANG II-mediated **TGF**-beta expression. Therefore, these drugs should not only be considered as antihypertensive medications, but should rather be viewed as renoprotective substances influencing renal remodeling by preventing local **TGF**-beta expression.

ANSWER 28 OF 40 CAPLUS COPYRIGHT 2003 ACS
idence suggests that transforming growth factor .beta.1 (**TGF** eta.1), a multifunctional cytokine, induces renal **extracellular** prodn. and glomerular hypertrophy. The effect of captopril estigated on the expression of **TGF**-.beta.1 mRNA in a rat chronic renal failure: 5/6 nephrectomy. Chronic renal disease

was induced by removal of the right kidney and ligation of 3 blood vessels supplying the left kidney. Sham-operated animals were used as controls. RNA was extd. from the viable remnant kidney of rats 1 day and 1 and 2 wk following 5/6 nephrectomy and from the kidneys of rats who underwent sham surgery. **TGF**-.beta.1 mRNA was induced within 24 h of partial nephrectomy, similar to that reported for early-onset genes. Subsequently, **TGF**-.beta.1 mRNA expression continued to increase over the next 2-4 wk. The upregulation of **TGF**-.beta.1 correlated with the degree of proteinuria. Both the increase in **TGF**-.beta.1 mRNA and proteinuria were abrogated by captopril treatment. In addn., no change in expression of ALK-5 or type II **TGF**-.beta. receptors following 5/6 nephrectomy was obsd. These data suggest that captopril may protect against development of glomerulosclerosis and proteinuria by reducing **TGF**-.beta.1 expression and hence matrix prodn.

L6 ANSWER 32 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI

AB Many of the pathophysiologic events associated with kidney disease are driven by angiotensin II. Irrespective of the etiology, many kidney diseases lead to tubulointerstitial inflammation, fibrosis and loss of renal function. Contributors to the process of tubulointerstitial fibrosis include monocyte/macrophage infiltration, the synthesis of profibrotic cytokines such as transforming growth factor beta 1 (**TGF**-beta 1), interstitial myofibroblast proliferation, and clusterin expression. These processes are ameliorated by angiotensin converting enzyme (ACE) inhibition. Blockade of the angiotensin II receptor (AT-1) impaired fibroblast proliferation, consequent differentiation into myofibroblasts, and the synthesis of **TGF**-beta 1, but did not prevent monocyte infiltration. AT-2 receptor blockade did not attenuate monocyte/macrophage infiltration, **TGF**-beta 1 synthesis or fibroblast proliferation but prevented the differentiation of fibroblasts into myofibroblasts and blocked clusterin expression. The nuclear factor-kappa B (NF-kappa B) family of transcription factors regulates genes involved in inflammation, proliferation and differentiation. ACE inhibition, AT-1 and AT-2 receptor blockade each differentially attenuated NF-kappa B isotype activation. The changes in NF-kappa B isotype may account for the variation seen in the pharmacologic effect of angiotensin II formation or action on the fibrotic process. When considering therapeutic options to prevent renal disease progression, one must be aware of the impact of transcription factors on the injured kidney and the consequent changes in cell infiltration, proliferation and differentiation.

L6 ANSWER 35 OF 40 MEDLINE DUPLICATE 14

AB We studied the effect of the angiotensin converting enzyme (**ACE**) **inhibitor**, quinapril, on the clinical and morphological lesions of a normotensive model of immune complex nephritis. Untreated rats developed massive nephrotic syndrome, intense cell proliferation and glomerular and tubulointerstitial lesions. In the renal cortex of nephritic rats there was a significant increase in gene expression of **TGF**-beta 1, fibronectin and collagens, and ACE activity. Systolic blood pressure remained normal with progression of the disease. Administration of quinapril for three weeks to animals with glomerular lesions (proteinuria 20 to 50 mg/day) avoided the development of intense proteinuria (79 +/- 28 vs. 589 +/- 73 mg/day, P < 0.001) and decreased cell proliferation, glomerulosclerosis, tubulointerstitial lesions, and inflammatory infiltrates. Cortical gene expression of **TGF**-beta 1 and matrix proteins was also diminished. ACE activity was inhibited by 68% in renal cortex. These results show that quinapril administration to normotensive rats with immune complex nephritis decreases proteinuria and glomerular and tubulointerstitial lesions, probably modulating the local angiotensin II generation and its effects on cell growth, **TGF** beta and matrix protein synthesis.

L6 ANSWER 39 OF 40 CAPLUS COPYRIGHT 2003 ACS

AB Smooth muscle cell (SMC) proliferation and formation of **extracellular matrix** in the intima of muscular arteries can lead to vascular stenosis in arteriosclerosis or after coronary angioplasty. The angiotensin-converting enzyme (**ACE**) **inhibitor** cilazapril strongly suppress this development of neointima. The beneficial effects on neointima formation persist for at least 8 wk after stopping treatment with cilazapril. Continuous treatment may have addnl. inhibitory effects during the late phases of vascular remodeling after injury. An **ACE inhibitor** of a different chem. class, captopril, reduced neointima formation as strongly as cilazapril (67 and 78%, resp.), but the calcium antagonist verapamil was not active as an inhibitor of neointima formation, despite similar lowering of blood pressure. Hydralazine and a new calcium antagonist, Ro 40-5967, partially suppressed neointima formation (36 and 33%, resp.). In vitro, neither cilazapril nor its active metabolite cilazaprilat had any effect on SMC proliferation in response to serum or blood platelet-derived growth factor (PDGF). The effects of angiotensin II (Ang II) and cilazaprilate on mRNA levels of several proteins potentially involved in regulating the SMC response were studied in cell cultures. Cilazaprilat did not alter the Ang II-induced increases of c-fos, c-myc, PDGF-A, and **TGF**-.beta., or the Ang II-induced decrease of PDGF .alpha.-receptor mRNAs. The converting enzyme has an important role in the proliferative response to vascular injury. Ang II may be a crit. regulatory factor in vivo during the response.

=> d 10 23 24 25 26 28 32 35 39

L6 ANSWER 10 OF 40 MEDLINE DUPLICATE 4
AN 2000507526 MEDLINE
DN 20510385 PubMed ID: 11054345
TI **ACE inhibitors** attenuate expression of renal transforming growth factor-beta1 in humans.
AU Shin G T; Kim S J; Ma K A; Kim H S; Kim D
CS Department of Nephrology, Ajou University School of Medicine, Suwon, South Korea.. gtshin@madang.ajou.ac.kr
SO AMERICAN JOURNAL OF KIDNEY DISEASES, (2000 Nov) 36 (5) 894-902. Journal code: 8110075. ISSN: 1523-6838.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200011
ED Entered STN: 20010322
Last Updated on STN: 20010521
Entered Medline: 20001109

L6 ANSWER 23 OF 40 CAPLUS COPYRIGHT 2003 ACS
AN 1999:6597 CAPLUS
DN 130:232278
TI Angiotensin converting enzyme inhibition reduces the expression of transforming growth factor-.beta.1 and type IV collagen in diabetic vasculopathy
AU Rumble, Jonathan R.; Gilbert, Richard E.; Cox, Alison; Wu, Leonard; Cooper, Mark E.
CS Department of Medicine, Austin & Repatriation Medical Centre, University of Melbourne, Heidelberg, VIC 3081, Australia
SO Journal of Hypertension (1998), 16(11), 1603-1609 CODEN: JOHYD3; ISSN: 0263-6352
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 24 OF 40 MEDLINE DUPLICATE 9
AN 1999062262 MEDLINE
DN 99062262 PubMed ID: 9844133
TI Targeting **TGF**-beta overexpression in renal disease: maximizing the antifibrotic action of angiotensin II blockade.
AU Peters H; Border W A; Noble N A
CS Division of Nephrology, University of Utah School of Medicine, Salt Lake City, Utah, USA.
NC DK 43609 (NIDDK)
DK 49342 (NIDDK)
DK 49374 (NIDDK)
SO KIDNEY INTERNATIONAL, (1998 Nov) 54 (5) 1570-80.
Journal code: 0323470. ISSN: 0085-2538.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199902
ED Entered STN: 19990223
Last Updated on STN: 19990223
Entered Medline: 19990211

L6 ANSWER 25 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 1998:171204 SCISEARCH
GA The Genuine Article (R) Number: YY259
TI Expression of transforming growth factor-beta 1 and type IV collagen in the renal tubulointerstitium in experimental diabetes - Effects of ACE inhibition
AU Gilbert R E (Reprint); Cox A; Wu L L; Allen T J; Hulthen U L; Jerums G; Cooper M E
CS UNIV MELBOURNE, ENDOCRINOL UNIT, AUSTIN & REPATRIAT MED CTR, DEPT MED, AUSTIN CAMPUS, STUDLEY RD, HEIDELBERG, VIC 3084, AUSTRALIA (Reprint)
CYA AUSTRALIA
SO DIABETES, (MAR 1998) Vol. 47, No. 3, pp. 414-422.
Publisher: AMER DIABETES ASSOC, 1660 DUKE ST, ALEXANDRIA, VA 22314.
ISSN: 0012-1797.
DT Article; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 51
*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

L6 ANSWER 26 OF 40 MEDLINE DUPLICATE 10
AN 1998184615 MEDLINE
DN 98184615 PubMed ID: 9525702
TI Link between angiotensin II and **TGF**-beta in the kidney.
AU Wolf G
CS Department of Medicine, University of Hamburg, Germany..
wolf@uke.uni-hamburg.de
SO MINERAL AND ELECTROLYTE METABOLISM, (1998) 24 (2-3) 174-80. Ref: 56
Journal code: 7802196. ISSN: 0378-0392.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199805
ED Entered STN: 19980514
Last Updated on STN: 19980514
Entered Medline: 19980501

L6 ANSWER 28 OF 40 CAPLUS COPYRIGHT 2003 ACS

AN 1998:436142 CAPLUS
DN 129:63504
TI Angiotensin-converting enzyme inhibition attenuates proteinuria and renal **TGF**-.beta.1 mRNA expression in rats with chronic renal disease
AU Ali, Shujath M.; Laping, Nicholas J.; Fredrickson, Todd A.; Contino, Lisa C.; Olson, Barbara A.; Anderson, Karen; Brooks, David P.
CS Department Renal Pharmacology, SmithKline Beecham Pharmaceuticals, King of Prussia, PA, 19406, USA
SO Pharmacology (1998), 57(1), 20-27
CODEN: PHMGBN; ISSN: 0031-7012
PB S. Karger AG
DT Journal
LA English

L6 ANSWER 32 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 97:905442 SCISEARCH
GA The Genuine Article (R) Number: YJ605
TI Comparative study of **ACE inhibitors** and angiotensin II receptor antagonists in interstitial scarring
AU Klahr S (Reprint); Morrissey J J
CS BARNES JEWISH HOSP, DEPT MED, 216 S KINGSHIGHWAY, ST LOUIS, MO 63110 (Reprint); WASHINGTON UNIV, SCH MED, DEPT MED, ST LOUIS, MO 63110; WASHINGTON UNIV, SCH MED, DEPT CELL BIOL & PHYSIOL, ST LOUIS, MO 63110
CYA USA
SO KIDNEY INTERNATIONAL, (DEC 1997) Supp. [63], pp. S111-S114.
Publisher: BLACKWELL SCIENCE INC, 350 MAIN ST, MALDEN, MA 02148.
ISSN: 0085-2538.
DT Article; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 19
*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

L6 ANSWER 35 OF 40 MEDLINE DUPLICATE 14
AN 96163237 MEDLINE
DN 96163237 PubMed ID: 8587237
TI ACE inhibition reduces proteinuria, glomerular lesions and **extracellular matrix** production in a normotensive rat model of immune complex nephritis.
AU Ruiz-Ortega M; Gonzalez S; Seron D; Condom E; Bustos C; Largo R; Gonzalez E; Ortiz A; Egido J
CS Renal Unit, Fundacion Jimenez Diaz, Universidad Autonoma, Madrid, Spain.
SO KIDNEY INTERNATIONAL, (1995 Dec) 48 (6) 1778-91.
Journal code: 0323470. ISSN: 0085-2538.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199603
ED Entered STN: 19960404
Last Updated on STN: 19960404
Entered Medline: 19960327

L6 ANSWER 39 OF 40 CAPLUS COPYRIGHT 2003 ACS
AN 1990:609229 CAPLUS
DN 113:209229
TI The proliferative response to vascular injury is suppressed by angiotensin-converting enzyme inhibition
AU Powell, Jerry S.; Mueller, Rita K. M.; Rouge, Marianne; Kuhn, Herbert; Hefti, Fridolin; Baumgartner, Hans R.
CS F. Hoffmann-La Roche Ltd., Basel, Switz.
SO Journal of Cardiovascular Pharmacology (1990), 16(Suppl. 4), S42-S49
CODEN: JCPCDT; ISSN: 0160-2446
DT Journal

LA English

=> d his

(FILE 'HOME' ENTERED AT 14:36:47 ON 19 MAY 2003)

FILE 'MEDLINE, CAPLUS, BIOTECHNO, SCISEARCH, BIOSIS' ENTERED AT 14:37:23
ON 19 MAY 2003
L1 108705 S TGF?
L2 1828 S L1 AND ECM
L3 10238 S L1 AND EXTRACELLULAR MATRIX
L4 10325 S L2 OR L3
L5 76 S L4 AND ACE INHIBITOR#
L6 40 DUP REM L5 (36 DUPLICATES REMOVED)

=> s l5 and tpa
L7 0 L5 AND TPA

=> s l4 and tpa
L8 77 L4 AND TPA

=> dup rem l8
PROCESSING COMPLETED FOR L8
L9 37 DUP REM L8 (40 DUPLICATES REMOVED)

=> d t 1-37
'T' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files. Refer to file specific help messages
or the STNGUIDE file for information on formats available in
individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):end

=> d ti 1-37

L9 ANSWER 1 OF 37 CAPLUS COPYRIGHT 2003 ACS
TI Role of membrane-bound heparin-binding epidermal growth factor-like growth factor (HB-EGF) in renal epithelial cell branching

L9 ANSWER 2 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Expression of glomerular plasminogen activator inhibitor type 1 in glomerulonephritis

L9 ANSWER 3 OF 37 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI The fibrinolytic receptor, annexin II, mediates epithelial-mesenchymal transformation in the developing avian heart.

L9 ANSWER 4 OF 37 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI Bradykinin reduces renal interstitial fibrosis by increasing **extracellular matrix** degradation.

L9 ANSWER 5 OF 37 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI Transforming growth factor (**TGF**)-beta1 inhibits human metalloelastase (MMP-12) through an activating protein (AP)-1 dependent, SMAD3 signaling pathway.

L9 ANSWER 6 OF 37 MEDLINE DUPLICATE 1
TI Increased expression of plasminogen activator and plasminogen activator inhibitor during liver fibrogenesis of rats: role of stellate cells.

L9 ANSWER 7 OF 37 MEDLINE DUPLICATE 2
TI Upregulation and spatial shift in the localization of the mannose 6-phosphate/insulin-like growth factor II receptor during radiation

enteropathy development in the rat.

L9 ANSWER 8 OF 37 MEDLINE DUPLICATE 3
TI Direct inhibitory effects of simvastatin on matrix accumulation in cultured murine mesangial cells.

L9 ANSWER 9 OF 37 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.
TI Direct inhibitory effects of simvastatin on matrix accumulation in cultured murine mesangial cells

L9 ANSWER 10 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Direct inhibitory effects of simvastatin on matrix accumulation in cultured murine mesangial cells

L9 ANSWER 11 OF 37 MEDLINE DUPLICATE 4
TI Immunocytochemical features of lens after cataract tissue--signalling molecules (growth factors, cytokines, other signalling molecules), cytoskeleton proteins, cellular and **extracellular matrix** proteins.

L9 ANSWER 12 OF 37 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI Direct inhibitory effects of simvastatin on matrix accumulation in cultured murine mesangial cells.

L9 ANSWER 13 OF 37 MEDLINE
TI Cytokine-induced changes in the ability of astrocytes to support migration of oligodendrocyte precursors and axon growth.

L9 ANSWER 14 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI Regulation of plasminogen activator inhibitor-1 mRNA accumulation by basic fibroblast growth factor and transforming growth factor-beta 1 in cultured rat astrocytes

L9 ANSWER 15 OF 37 CAPLUS COPYRIGHT 2003 ACS
TI Regulation of tissue plasminogen activator production in cultured human fetal mesangial cells

L9 ANSWER 16 OF 37 MEDLINE DUPLICATE 5
TI Growth factor and cytokine modulation of trabecular meshwork matrix metalloproteinase and TIMP expression.

L9 ANSWER 17 OF 37 MEDLINE DUPLICATE 6
TI Humatrix, a novel myoepithelial matrical gel with unique biochemical and biological properties.

L9 ANSWER 18 OF 37 MEDLINE DUPLICATE 7
TI PAI-1 secretion and matrix deposition in human peritoneal mesothelial cell cultures: transcriptional regulation by **TGF**-beta 1.

L9 ANSWER 19 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI SV40-transformation of embryonic human diploid fibroblasts results in multiple molecular changes

L9 ANSWER 20 OF 37 MEDLINE DUPLICATE 8
TI Increased expression of **extracellular matrix** proteins and decreased expression of matrix proteases after serial passage of glomerular mesangial cells.

L9 ANSWER 21 OF 37 MEDLINE DUPLICATE 9
TI Effect of transforming growth factor-beta on plasminogen activator production of cultured human uveal melanoma cells.

L9 ANSWER 22 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI STRUCTURE, FUNCTION AND REGULATION OF LIPOPROTEIN(A)

L9 ANSWER 23 OF 37 MEDLINE DUPLICATE 10
TI Induction of plasminogen activator inhibitor type 1 in murine lupus-like glomerulonephritis.

L9 ANSWER 24 OF 37 MEDLINE DUPLICATE 11
TI Concerted action of **TGF**-beta 1 and its type II receptor in control of epidermal homeostasis in transgenic mice.

L9 ANSWER 25 OF 37 MEDLINE DUPLICATE 12
TI Prostaglandin E2 regulates production of plasminogen activator isoenzymes, urokinase receptor, and plasminogen activator inhibitor-1 in primary cultures of rat calvarial osteoblasts.

L9 ANSWER 26 OF 37 CAPLUS COPYRIGHT 2003 ACS
TI Expression of tissue-type plasminogen activator and its inhibitor couples with development of capillary network by human microvascular endothelial cells on Matrigel

L9 ANSWER 27 OF 37 MEDLINE
TI Effects of complete and incomplete tumor promoters on hair growth, angiogenesis, and tenascin expression in the skin of NMRI mice.

L9 ANSWER 28 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI PROTEASES AND INVASION BY METASTATIC TUMOR-CELLS - CLINICAL IMPLICATIONS FOR PROSTATE-CANCER

L9 ANSWER 29 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI IMMUNOHISTOCHEMICAL INSIGHTS INTO SICKLE-CELL RETINOPATHY

L9 ANSWER 30 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI INDUCTION OF MEMBRANE RUFFLING BY GROWTH-FACTORS IN MORPHOLOGICALLY **TPA**-RESISTANT BALB/C3T3 TR4 CELLS

L9 ANSWER 31 OF 37 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.
TI Interleukin-1.beta. and transforming growth factor-.alpha./epidermal growth factor induce expression of M(r) 95,000 type IV collagenase/gelatinase and interstitial fibroblast-type collagenase by rat mucosal keratinocytes

L9 ANSWER 32 OF 37 MEDLINE DUPLICATE 13
TI Induction of metalloproteinase activity in human T-lymphocytes.

L9 ANSWER 33 OF 37 MEDLINE DUPLICATE 14
TI Transforming growth factor beta as a neuronoglial signal during peripheral nervous system response to injury.

L9 ANSWER 34 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI AUTOCRINE SECRETION OF TRANSFORMING GROWTH-FACTOR-BETA IN CULTURED RAT MESANGIAL CELLS

L9 ANSWER 35 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI ALTERATIONS IN MESSENGER-RNA LEVELS FOR GROWTH-RELATED GENES AFTER TRANSPLANTATION INTO CASTRATED HOSTS IN ONCOGENE-INDUCED CLONAL MOUSE PROSTATE CARCINOMA

L9 ANSWER 36 OF 37 CAPLUS COPYRIGHT 2003 ACS
TI Transforming growth factor-.beta.1 up-regulates type IV collagenase expression in cultured human keratinocytes

L9 ANSWER 37 OF 37 MEDLINE DUPLICATE 15
TI Cell type-specific control of human neuronectin secretion by polypeptide mediators and phorbol ester.

=> d 36 31 30 23 20 4 2

L9 ANSWER 36 OF 37 CAPLUS COPYRIGHT 2003 ACS
AN 1991:507031 CAPLUS
DN 115:107031
TI Transforming growth factor-.beta.1 up-regulates type IV collagenase expression in cultured human keratinocytes
AU Salo, Tuula; Lyons, J. Guy; Rahemtulla, Firoz; Birkedal-Hansen, Henning; Larjava, Hannu
CS Dep. Oral Surg., Univ. Oulu, Oulu, SF-90220, Finland
SO Journal of Biological Chemistry (1991), 266(18), 11436-41
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English

L9 ANSWER 31 OF 37 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.
AN 1993:23273871 BIOTECHNO
TI Interleukin-1.beta. and transforming growth factor-.alpha./epidermal growth factor induce expression of M(r) 95,000 type IV collagenase/gelatinase and interstitial fibroblast-type collagenase by rat mucosal keratinocytes
AU Lyons J.G.; Birkedal-Hansen B.; Pierson M.C.; Whitelock J.M.; Birkedal-Hansen H.
CS Dept. of Oral Biology, Univ. of Alabama School of Dentistry, SDB Box 54,Birmingham, AL 35294, United States.
SO Journal of Biological Chemistry, (1993), 268/25 (19143-19151)
CODEN: JBCHA3 ISSN: 0021-9258
DT Journal; Article
CY United States
LA English
SL English

L9 ANSWER 30 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 94:381608 SCISEARCH
GA The Genuine Article (R) Number: NR016
TI INDUCTION OF MEMBRANE RUFFLING BY GROWTH-FACTORS IN MORPHOLOGICALLY **TPA**-RESISTANT BALB/C3T3 TR4 CELLS
AU ENOMOTO T (Reprint); ASANO Y
CS KOBE UNIV, SCH MED, DEPT RADIAT BIOPHYS & GENET, CHUO KU, KUSUNOKI 7-5-1, KOBE 650, JAPAN (Reprint); OSAKA UNIV, MICROBIAL DIS RES INST, DEPT ONCOGENE RES, SUITA, OSAKA 565, JAPAN
CYA JAPAN
SO CELL STRUCTURE AND FUNCTION, (APR 1994) Vol. 19, No. 2, pp. 89-96.
ISSN: 0386-7196.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 32
*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

L9 ANSWER 23 OF 37 MEDLINE DUPLICATE 10
AN 96130535 MEDLINE
DN 96130535 PubMed ID: 8544402
TI Induction of plasminogen activator inhibitor type 1 in murine lupus-like glomerulonephritis.
AU Moll S; Menoud P A; Fulpius T; Pastore Y; Takahashi S; Fossati L; Vassalli J D; Sappino A P; Schifferli J A; Izui S
CS Department of Pathology, University of Geneva Medical School, Switzerland.
SO KIDNEY INTERNATIONAL, (1995 Nov) 48 (5) 1459-68.
Journal code: 0323470. ISSN: 0085-2538.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals
EM 199602
ED Entered STN: 19960227
Last Updated on STN: 19960227
Entered Medline: 19960214

L9 ANSWER 20 OF 37 MEDLINE DUPLICATE 8
AN 97081970 MEDLINE
DN 97081970 PubMed ID: 8923213
TI Increased expression of **extracellular matrix** proteins and decreased expression of matrix proteases after serial passage of glomerular mesangial cells.
AU Schnaper H W; Kopp J B; Poncelet A C; Hubchak S C; Stetler-Stevenson W G; Klotman P E; Kleinman H K
CS Department of Pediatrics, Northwestern University Medical School, Chicago, IL 60611-3008, USA.
NC R01-DK49362 (NIDDK)
SO JOURNAL OF CELL SCIENCE, (1996 Oct) 109 ( Pt 10) 2521-8.
Journal code: 0052457. ISSN: 0021-9533.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199705
ED Entered STN: 19970523
Last Updated on STN: 19980206
Entered Medline: 19970515

L9 ANSWER 4 OF 37 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 2002:320800 BIOSIS
DN PREV200200320800
TI Bradykinin reduces renal interstitial fibrosis by increasing **extracellular matrix** degradation.
AU Schanstra, Joost P. (1); Drogoz, Pascale (1); Desmond, Laurence (1); Calise, Denis (1); Neau, Eric (1); Girolami, Jean-Pierre (1); Bascands, Jean-Loup (1)
CS (1) U388, INSERM, Toulouse Cedex 4 France
SO Journal of the American Society of Nephrology, (September, 2001) Vol. 12, No. Program and Abstract Issue, pp. 716A. http://www.jasn.org/. print.
Meeting Info.: ASN (American Society of Nephrology)/ISN (International Society of Nephrology) World Congress of Nephrology San Francisco, CA, USA October 10-17, 2001
ISSN: 1046-6673.
DT Conference
LA English

L9 ANSWER 2 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 2002:390963 SCISEARCH
GA The Genuine Article (R) Number: 538VD
TI Expression of glomerular plasminogen activator inhibitor type 1 in glomerulonephritis
AU Hamano K; Iwano M (Reprint); Akai Y; Sato H; Kubo A; Nishitani Y; Uyama H; Yoshida Y; Miyazaki M; Shiiki H; Kohno S; Dohi K
CS Nara Med Univ, Dept Internal Med 1, 840 Shijo, Kashihara, Nara 6348522, Japan (Reprint); Nara Med Univ, Dept Internal Med 1, Kashihara, Nara 6348522, Japan; Nagasaki Univ, Sch Med, Dept Internal Med 2, Nagasaki 852, Japan
CYA Japan
SO AMERICAN JOURNAL OF KIDNEY DISEASES, (APR 2002) Vol. 39, No. 4, pp. 695-705.
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399 USA.
ISSN: 0272-6386.
DT Article; Journal

LA English
REC Reference Count: 55
*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

=> d ab 36 31 30 23 20 4 2

L9 ANSWER 36 OF 37 CAPLUS COPYRIGHT 2003 ACS
AB During the wound healing process lysis of basement membranes precedes keratinocyte migration into the wound bed. Whether this degrdn. of basement membranes could be regulated by transforming growth factor-.beta.1 (**TGF**-.beta.1), which is known to accelerate wound healing in vivo, was studied in vivo. Transforming growth factor-.beta.1 increased the expression of both 92- and 72-kDa type IV collagenases (gelatinases) in cultured human mucosal and dermal keratinocytes. The 92-kDa enzyme predominated in both unstimulated and stimulated cultures. The 92-kDa form was stimulated over 5-fold, and the other form by a factor of 2-3. This increase in the synthesis of type IV collagenases was assocd. with a marked increase in the mRNA levels of these enzymes as well. The induction of the 92-kDa enzyme was similar in culture medium contg. either 0.15 or 1.2 mM CaCl2. Rat mucosal keratinocytes secreted only 92-kDa type IV collagenase, the secretion of which was not regulated by **TGF**-.beta.1. Also, **TGF**-.beta.1 did not cause any significant induction (max. about 1.2-fold) of either type IV collagenase in human gingival fibroblasts. The induction levels of both collagenases in human keratinocytes were independent of the type of the **extracellular matrix** the cells were grown on. However, the basement membrane matrix (Matrigel) activated about half of the 92-kDa type to its 84-kDa active form. The data suggest that **TGF**-.beta.1 has a specific function in up-regulating the expression of type IV collagenases in human keratinocytes, offering a possible explanation of how keratinocytes detach from basement membranes prior to the migration over the wound bed.

L9 ANSWER 31 OF 37 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.
AB Rat mucosal keratinocytes serially propagated under permanently serum-free conditions responded to interleukin (IL)-I.beta./IL-.alpha. and to transforming growth factor (**TGF**)-.alpha./epidermal growth factor (EGF) (as well as to 12-O- tetradecanoylphorbol-13-acetate (**TPA**)) by upregulation of M(r) 95,000 gelatinase (MMP-9) (M(r) 95K GL) and fibroblast-type collagenase (MMP-1) (FIB-CL), whereas control cells expressed barely detectable levels of either of these enzymes. The cells secreted 8-10 .mu.g/10.sup.6 cells/day (M(r) 95K GL) and 2-3 .mu.g/10.sup.6 cells/day (FIB-CL) of enzyme protein for at least 24 h when maximally induced. This level was attained only after a 24-h lag period, and the earliest emergence of enzyme protein in the culture medium required 10- 14 h. IL-1.beta. was by far the most potent cytokine with maximal effect already at 10.sup.-.sup.1.sup.0 M, whereas IL-1.alpha., **TGF**-.alpha., and EGF required 20-100-fold higher concentrations. Pretreatment of the cells with **TPA** (10.sup.-.sup.7 M) abolished the subsequent response to IL-1.beta., **TGF**-.alpha., and EGF and at the same time resulted in >90% reduction of cytosolic protein kinase C activity. Surprisingly, staurosporine, a potent kinase inhibitor, not only failed to block growth factor/cytokine responses but itself stimulated expression of the enzymes at a magnitude comparable to **TPA**. The inducing effect of **TGF**-.alpha./EGF was downregulated by 70-85% by 10.sup.-.sup.7 M dexamethasone. Dexamethasone was less effective in ablating the IL-1.beta. response yielding 60% reduction M(r) 95K GL and little or no reduction of FIB-CL. Dexamethasone also failed to block the **TPA** response.

L9 ANSWER 30 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AB To investigate the biological characteristics of a Balb/c3T3 variant

TR4 clone which is morphologically resistant to **TPA** and hypersensitive to v-src induced metastasis, we compared the responsiveness of the variant and its parent cells to growth factor-induced membrane ruffling. When the confluent cells were stimulated with PDGF, membrane ruffling was rapidly induced in TR4 but not in the parent cell cultures. In TR4 cells, membrane ruffling was observed under a phase-contrast microscope within 2 min after the addition of PDGF, reaching the maximum 5 min later and thereafter decreased gradually to the control level. There were no apparent differences in I-125-PDGF binding kinetics between TR4 and parent cells. Similar membrane ruffling was induced by other growth factors such as insulin, IGF-I, acidic or basic FGF but not by EGF or alpha- and beta-**TGF**, only in TR4 cells. When TR4 cells were incubated with **TPA** just before stimulation with these growth factors, growth factor-induced membrane ruffling was completely inhibited. Also, 5 out of 6 clones of stable fusion cells between TR4 and parent cells showed the parental type of responses to **TPA** and growth factors, indicating that the TR4 phenotype is recessive. These results suggest that the variant TR4 cells may acquire the genetic and recessive alteration of a cellular factor which is responsible for the regulation of growth factor-mediated membrane ruffling and that this genetic alteration occurs at a common step downstream of growth factor-mediated cascades, rather than at their receptor level.

L9 ANSWER 23 OF 37 MEDLINE DUPLICATE 10

AB Three major components of the plasminogen activators (PA)/plasmin system are synthesized physiologically in glomeruli, and can be involved in glomerular proteolysis and **extracellular matrix** metabolism: tissue-type PA (**tPA**), urokinase (uPA) and PA inhibitor type 1 (PAI-1). To explore the possible role of a dysregulation of the plasmin protease system in the development and progression of lupus-like glomerulonephritis, we studied the expression of the renal plasmin protease components during the course of the disease, either acute, induced by IgG3 monoclonal cryoglobulins, or chronic, occurring spontaneously in three different lupus-prone mice: (NZBxNZW)F1, BXSB and MRL-lpr/lpr. RNase protection assays and in situ hybridizations revealed a marked glomerular induction of PAI-1 mRNA abundance without any significant changes in renal **tPA** and uPA mRNA levels in the two different types of lupus-like glomerulonephritis. The overexpression of PAI-1 mRNA occurred in parallel with a significant decrease in glomerular **tPA**-catalyzed enzymatic activity as determined by zymographic analysis. In addition, a concomitant increase in glomerular expression of transforming growth factor beta 1 (**TGF**-beta 1) mRNA was observed. The demonstration of a close correlation between the PAI-1 and **TGF**-beta 1 mRNA levels and the severity of lupus-like glomerular lesions suggests that a pertubation of the glomerular PA/PAI balance, resulting from a marked **TGF**-beta 1-mediated induction of PAI-1 gene expression, plays an important role in the progression of lupus-like glomerular lesions, leading to glomerulosclerosis.

L9 ANSWER 20 OF 37 MEDLINE DUPLICATE 8

AB The cellular events causing pathological **extracellular matrix** (**ECM**) accumulation in vivo are not well understood. Prolonged serial passage of several cell types in culture leads to increased production of **extracellular matrix** (**ECM**) proteins, but the mechanism for these putative fibrotic changes is not known. Here, human fetal glomerular mesangial cells were subjected to serial passage (P) in culture and the expression of **ECM** proteins, proteases and protease inhibitors was comprehensively evaluated. From P11 through P14, a series of phenotypic changes occurred. Steady-state expression of mRNA for alpha 1 chains of type III and type IV (but not type I) collagen, and for laminin beta 1 and gamma 1, increased 2- to 8-fold, while expression of mRNA for interstitial collagenase (MMP-1) and gelatinase A (MMP-2) virtually ceased. Expression of tissue-type plasminogen activator (**tPA**) mRNA also decreased

markedly. Expression of mRNA for the tissue inhibitor of metalloproteinases (TIMP)-1, and of the smaller of two mRNA species for the PA inhibitor PAI-1, ceased by P14. There was a switch in expression of the two species of TIMP-2 mRNA: whereas the ratio of signal intensity comparing the 3.5 kb mRNA species to the 1.0 kb species was 5:1 up to P11, it was reversed (1:5) at P14 and later. Serial passage also led to changes in protein expression, with increased type IV collagen and laminin, but decreased interstitial collagenase and gelatinase A. The cells showed a progressive increase in staining for type IV collagen. These findings define the appearance of a matrix-accumulating phenotype in later-passage mesangial cells. Matrix expansion in vivo has been associated with increased transforming growth factor (**TGF**)-beta synthesis; the cells were found to show at least 5-fold increased expression of **TGF**-beta 1 mRNA from P8 to P16. However, treatment of P9 or P10 cells with graded doses of **TGF**-beta 1 increased expression of both collagen IV and gelatinase A mRNA and did not alter the ratio of signal intensity for TIMP-2 mRNA species. Thus, assumption of a matrix-accumulating phenotype by these cultured fetal glomerular mesangial cells is not accelerated by exogenous **TGF** -beta. These data describe an in vitro model of mesangial cell matrix turnover in which matrix accumulation could result from a concerted increase in **ECM** synthesis and decrease in **ECM** degradation.

L9 ANSWER 4 OF 37 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

L9 ANSWER 2 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI

AB Plasminogen activator inhibitor type 1 (PAI-1) and tissue plasminogen activator (**tPA**) are the major regulators of plasmin generation. Glomerular PAI-1/**tPA** balance is involved in **extracellular matrix** turnover, as well as fibrin deposition in glomerull. Renal biopsy specimens were obtained from 80 patients with either primary or secondary glomerulonephritis (10 patients, minimal change nephrotic syndrome; 6 patients, focal segmental glomerulosclerosis [FSGS]; 10 patients, membranous nephropathy [MN]; 24 patients, mesangial proliferative glomerulonephritis; 15 patients, lupus nephritis; 14 patients, diabetic nephropathy; and I patient, membranoproliferative glomerulonephritis). We quantified glomerular PAI-1 and **tPA** messenger RNA (mRNA) by competitive polymerase chain reaction. We also determined PAI-1 mRNA localization by In situ hybridization. Glomerular PAI-1 mRNA levels in patients with FSGS and MN were significantly greater than those of controls. There was a sixfold increase in PAI-1-**tPA** mRNA ratio in patients with MN compared with the control group. In addition, glomerular PAI-1 mRNA level correlated with level of proteinuria. Conversely, there was no difference in **tPA** mRNA levels among types of glomerulonephritis. These results suggest that suppressed glomerular fibrinolytic and proteolytic activity may be associated with the pathogenesis of glomerulonephritis, especially in FSGS and MN. (C) 2002 by the National Kidney Foundation, Inc.

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(FILE 'HOME' ENTERED AT 14:36:47 ON 19 MAY 2003)

FILE 'MEDLINE, CAPLUS, BIOTECHNO, SCISEARCH, BIOSIS' ENTERED AT 14:37:23 ON 19 MAY 2003

L1 108705 S TGF?
L2 1828 S L1 AND ECM
L3 10238 S L1 AND EXTRACELLULAR MATRIX
L4 10325 S L2 OR L3
L5 76 S L4 AND ACE INHIBITOR#
L6 40 DUP REM L5 (36 DUPLICATES REMOVED)

L7 0 S L5 AND TPA
L8 77 S L4 AND TPA
L9 37 DUP REM L8 (40 DUPLICATES REMOVED)

=> s l5 and protease
L10 5 L5 AND PROTEASE

=> d 1-5 ab

L10 ANSWER 1 OF 5 MEDLINE
AB BACKGROUND: Overproduction of transforming growth factor-beta (**TGF**-beta) is a key mediator of **extracellular matrix** accumulation in fibrotic diseases. We hypothesized that the degree of reduction of pathological **TGF**-beta expression can be used as a novel index of the antifibrotic potential of angiotensin II (Ang II) blockade in renal disease. METHODS: One day after induction of Thy 1.1 glomerulonephritis, rats were treated with increasing doses of the Ang I converting enzyme (**ACE**) **inhibitor** enalapril and/or the Ang II receptor blocker losartan in the drinking water. Six days after disease induction the therapeutic effect on glomerular **TGF**-beta overexpression was evaluated. RESULTS: Both enalapril and losartan reduced **TGF**-beta overproduction in a dose-dependent manner, showing a moderate reduction at doses known to control blood pressure in renal forms of hypertension. A maximal reduction in **TGF**-beta expression of approximately 45% was seen for both drugs starting at 100 mg/liter enalapril and 500 mg/liter losartan, with no further reduction at doses of enalapril up to 1000 mg/liter or losartan up to 2500 mg/liter. Co-treatment with both drugs was not superior to single therapy. Consistent with our hypothesis that reduction in **TGF**-beta expression is a valid target, other disease measures, including glomerular matrix accumulation, glomerular production and mRNA expression of the matrix protein fibronectin and the **protease** inhibitor plasminogen-activator-inhibitor type 1 (PAI-1) closely followed **TGF**-beta expression. CONCLUSIONS: The data suggest that these therapies act through very similar pathways and that, in order to more effectively treat renal fibrosis, these drugs must be combined with other drugs that act by different mechanisms.

L10 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2003 ACS
AB Overprodn. of transforming growth factor-.beta. (**TGF**-.beta.) is a key mediator of **extracellular matrix** accumulation in fibrotic diseases. We hypothesized that the degree of redn. of pathol. **TGF**-.beta. expression can be used as a novel index of the antifibrotic potential of angiotensin II (Ang II) blockade in renal disease. One day after induction of Thy 1.1 glomerulonephritis, rats were treated with increasing doses of the Ang I converting enzyme (**ACE**) **inhibitor** enalapril and/or the Ang II receptor blocker losartan in the drinking water. Six days after disease induction the therapeutic effect on glomerular **TGF**-.beta. overexpression was evaluated. Both enalapril and losartan reduced **TGF**-.beta. overprodn. in a dose-dependent manner, showing a moderate redn. at doses known to control blood pressure in renal forms of hypertension. A maximal redn. in **TGF**-.beta. expression of approx. 45% was seen for both drugs starting at 100 mg/L enalapril and 500 mg/L losartan, with no further redn. at doses of enalapril up to 1000 mg/L or losartan up to 2500 mg/L. Co-treatment with both drugs was not superior to single therapy. Consistent with our hypothesis that redn. in **TGF**-.beta. expression is a valid target, other disease measures, including glomerular matrix accumulation, glomerular prodn. and mRNA expression of the matrix protein fibronectin and the **protease** inhibitor plasminogen-activator-inhibitor type 1 (PAI-1) closely followed **TGF**-.beta. expression. The data suggest that these therapies act through very similar pathways and that, in order to more effectively treat renal fibrosis, these drugs must be combined with other drugs that act by

different mechanisms.

L10 ANSWER 3 OF 5 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.
AB Background. Overproduction of transforming growth factor-.beta. ( **TGF**-.beta.) is a key mediator of **extracellular matrix** accumulation in fibrotic diseases. We hypothesized that the degree of reduction of pathological **TGF**-.beta. expression can be used as a novel index of the antifibrotic potential of angiotensin II (Ang II) blockade in renal disease. Methods. One day after induction of Thy 1.1 glomerulonephritis, rats were treated with increasing doses of the Ang I converting enzyme (**ACE**) **inhibitor** enalapril and/or the Ang II receptor blocker losartan in the drinking water. Six days after disease induction the therapeutic effect on glomerular **TGF**-.beta. overexpression was evaluated. Results. Both enalapril and losartan reduced **TGF**-.beta. overproduction in a dose-dependent manner, showing a moderate reduction at doses known to control blood pressure in renal forms of hypertension. A maximal reduction in **TGF**-.beta. expression of approximately 45% was seen for both drugs starting at 100 mg/liter enalapril and 500 mg/liter losartan, with no further reduction at doses of enalapril up to 1000 mg/liter or losartan up to 2500 mg/liter. Co- treatment with both drugs was not superior to single therapy. Consistent with our hypothesis that reduction in **TGF**-.beta. expression is a valid target, other disease measures, including glomerular matrix accumulation, glomerular production and mRNA expression of the matrix protein fibronectin and the **protease** inhibitor plasminogen-activator-inhibitor type 1 (PAI-1) closely followed **TGF**-.beta. expression. Conclusions. The data suggest that these therapies act through very similar pathways and that, in order to more effectively treat renal fibrosis, these drugs must be combined with other drugs that act by different mechanisms.

L10 ANSWER 4 OF 5 SCISEARCH COPYRIGHT 2003 THOMSON ISI
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Conclusions. The data suggest that these therapies act through very similar pathways and that, in order to more effectively treat renal fibrosis, these drugs must be combined with other drugs that act by different mechanisms.

L10 ANSWER 5 OF 5 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AB Background. Overproduction of transforming growth factor-beta (**TGF**

-beta) is a key mediator of **extracellular matrix** accumulation in fibrotic diseases. We hypothesized that the degree of reduction of pathological **TGF**-beta expression can be used as a novel index of the antifibrotic potential of angiotensin II (Ang II) blockade in renal disease. Methods. One day after induction of Thy 1.1 glomerulonephritis, rats were treated with increasing doses of the Ang I converting enzyme (**ACE**) **inhibitor** enalapril and/or the Ang II receptor blocker losartan in the drinking water. Six days after disease induction the therapeutic effect on glomerular **TGF**-beta overexpression was evaluated. Results. Both enalapril and losartan reduced **TGF**-beta overproduction in a dose-dependent manner, showing a moderate reduction at doses known to control blood pressure in renal forms of hypertension. A maximal reduction in **TGF**-beta expression of approximately 45% was seen for both drugs starting at 100 mg/liter enalapril and 500 mg/liter losartan, with no further reduction at doses of enalapril up to 1000 mg/liter or losartan up to 2500 mg/liter. Co-treatment with both drugs was not superior to single therapy. Consistent with our hypothesis that reduction in **TGF**-beta expression is a valid target, other disease measures, including glomerular matrix accumulation, glomerular production and mRNA expression of the matrix protein fibronectin and the **protease** inhibitor plasminogen-activator-inhibitor type 1 (PAI-1) closely followed **TGF**-beta expression. Conclusions. The data suggest that these therapies act through very similar pathways and that, in order to more effectively treat renal fibrosis, these drugs must be combined with other drugs that act by different mechanisms.

=> d

L10 ANSWER 1 OF 5 MEDLINE
AN 1999062262 MEDLINE
DN 99062262 PubMed ID: 9844133
TI Targeting **TGF**-beta overexpression in renal disease: maximizing the antifibrotic action of angiotensin II blockade.
AU Peters H; Border W A; Noble N A
CS Division of Nephrology, University of Utah School of Medicine, Salt Lake City, Utah, USA.
NC DK 43609 (NIDDK)
DK 49342 (NIDDK)
DK 49374 (NIDDK)
SO KIDNEY INTERNATIONAL, (1998 Nov) 54 (5) 1570-80.
Journal code: 0323470. ISSN: 0085-2538.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199902
ED Entered STN: 19990223
Last Updated on STN: 19990223
Entered Medline: 19990211

L6 ANSWER 10 OF 40 MEDLINE DUPLICATE 4
AN 2000507526 MEDLINE
DN 20510385 PubMed ID: 11054345
TI ACE inhibitors attenuate expression of renal transforming growth factor-beta1 in humans.
AU Shin G T; Kim S J; Ma K A; Kim H S; Kim D
CS Department of Nephrology, Ajou University School of Medicine, Suwon, South Korea.. gtshin@madang.ajou.ac.kr
SO AMERICAN JOURNAL OF KIDNEY DISEASES, (2000 Nov) 36 (5) 894-902. Journal code: 8110075. ISSN: 1523-6838.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200011
ED Entered STN: 20010322
Last Updated on STN: 20010521
Entered Medline: 20001109

L6 ANSWER 23 OF 40 CAPLUS COPYRIGHT 2003 ACS
AN 1999:6597 CAPLUS
DN 130:232278
TI Angiotensin converting enzyme inhibition reduces the expression of transforming growth factor-.beta.1 and type IV collagen in diabetic vasculopathy
AU Rumble, Jonathan R.; Gilbert, Richard E.; Cox, Alison; Wu, Leonard; Cooper, Mark E.
CS Department of Medicine, Austin & Repatriation Medical Centre, University of Melbourne, Heidelberg, VIC 3081, Australia
SO Journal of Hypertension (1998), 16(11), 1603-1609
CODEN: JOHYD3; ISSN: 0263-6352
PB Lippincott Williams & Wilkins
DT Journal
LA English
RE.CNT 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 24 OF 40 MEDLINE DUPLICATE 9
AN 1999062262 MEDLINE
DN 99062262 PubMed ID: 9844133
TI Targeting TGF-beta overexpression in renal disease: maximizing the antifibrotic action of angiotensin II blockade.
AU Peters H; Border W A; Noble N A
CS Division of Nephrology, University of Utah School of Medicine, Salt Lake City, Utah, USA.
NC DK 43609 (NIDDK)
DK 49342 (NIDDK)
DK 49374 (NIDDK)
SO KIDNEY INTERNATIONAL, (1998 Nov) 54 (5) 1570-80. Journal code: 0323470. ISSN: 0085-2538.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199902
ED Entered STN: 19990223
Last Updated on STN: 19990223
Entered Medline: 19990211

L6 ANSWER 25 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 1998:171204 SCISEARCH
GA The Genuine Article (R) Number: YY259
TI Expression of transforming growth factor-beta 1 and type IV collagen in the renal tubulointerstitium in experimental diabetes - Effects of ACE inhibition
AU Gilbert R E (Reprint); Cox A; Wu L L; Allen T J; Hulthen U L; Jerums G; Cooper M E
CS UNIV MELBOURNE, ENDOCRINOL UNIT, AUSTIN & REPATRIAT MED CTR, DEPT MED, AUSTIN CAMPUS, STUDLEY RD, HEIDELBERG, VIC 3084, AUSTRALIA (Reprint)
CYA AUSTRALIA
SO DIABETES, (MAR 1998) Vol. 47, No. 3, pp. 414-422.
Publisher: AMER DIABETES ASSOC, 1660 DUKE ST, ALEXANDRIA, VA 22314.
ISSN: 0012-1797.
DT Article; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 51
*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

L6 ANSWER 26 OF 40 MEDLINE DUPLICATE 10
AN 1998184615 MEDLINE
DN 98184615 PubMed ID: 9525702
TI Link between angiotensin II and TGF-beta in the kidney.
AU Wolf G
CS Department of Medicine, University of Hamburg, Germany..
wolf@uke.uni-hamburg.de
SO MINERAL AND ELECTROLYTE METABOLISM, (1998) 24 (2-3) 174-80. Ref: 56
Journal code: 7802196. ISSN: 0378-0392.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199805
ED Entered STN: 19980514
Last Updated on STN: 19980514
Entered Medline: 19980501

L6 ANSWER 28 OF 40 CAPLUS COPYRIGHT 2003 ACS
AN 1998:436142 CAPLUS
DN 129:63504
TI Angiotensin-converting enzyme inhibition attenuates proteinuria and renal TGF-.beta.1 mRNA expression in rats with chronic renal disease
AU Ali, Shujath M.; Laping, Nicholas J.; Fredrickson, Todd A.; Contino, Lisa C.; Olson, Barbara A.; Anderson, Karen; Brooks, David P.
CS Department Renal Pharmacology, SmithKline Beecham Pharmaceuticals, King of Prussia, PA, 19406, USA
SO Pharmacology (1998), 57(1), 20-27
CODEN: PHMGBN; ISSN: 0031-7012
PB S. Karger AG
DT Journal
LA English

L6 ANSWER 32 OF 40 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 97:905442 SCISEARCH
GA The Genuine Article (R) Number: YJ605
TI Comparative study of ACE inhibitors and angiotensin II receptor antagonists in interstitial scarring
AU Klahr S (Reprint); Morrissey J J
CS BARNES JEWISH HOSP, DEPT MED, 216 S KINGSHIGHWAY, ST LOUIS, MO 63110 (Reprint); WASHINGTON UNIV, SCH MED, DEPT MED, ST LOUIS, MO 63110; WASHINGTON UNIV, SCH MED, DEPT CELL BIOL & PHYSIOL, ST LOUIS, MO 63110

CYA USA
SO KIDNEY INTERNATIONAL, (DEC 1997) Supp. [63], pp. S111-S114.
Publisher: BLACKWELL SCIENCE INC, 350 MAIN ST, MALDEN, MA 02148.
ISSN: 0085-2538.
DT Article; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 19
*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

L6 ANSWER 35 OF 40 MEDLINE DUPLICATE 14
AN 96163237 MEDLINE
DN 96163237 PubMed ID: 8587237
TI ACE inhibition reduces proteinuria, glomerular lesions and extracellular matrix production in a normotensive rat model of immune complex nephritis.
AU Ruiz-Ortega M; Gonzalez S; Seron D; Condom E; Bustos C; Largo R; Gonzalez E; Ortiz A; Egido J
CS Renal Unit, Fundacion Jimenez Diaz, Universidad Autonoma, Madrid, Spain.
SO KIDNEY INTERNATIONAL, (1995 Dec) 48 (6) 1778-91.
Journal code: 0323470. ISSN: 0085-2538.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199603
ED Entered STN: 19960404
Last Updated on STN: 19960404
Entered Medline: 19960327

L6 ANSWER 39 OF 40 CAPLUS COPYRIGHT 2003 ACS
AN 1990:609229 CAPLUS
DN 113:209229
TI The proliferative response to vascular injury is suppressed by angiotensin-converting enzyme inhibition
AU Powell, Jerry S.; Mueller, Rita K. M.; Rouge, Marianne; Kuhn, Herbert; Hefti, Fridolin; Baumgartner, Hans R.
CS F. Hoffmann-La Roche Ltd., Basel, Switz.
SO Journal of Cardiovascular Pharmacology (1990), 16(Suppl. 4), S42-S49
CODEN: JCPCDT; ISSN: 0160-2446
DT Journal
LA English

ANSWER 36 OF 37 CAPLUS COPYRIGHT 2003 ACS
AN 1991:507031 CAPLUS
DN 115:107031
TI Transforming growth factor-.beta.1 up-regulates type IV collagenase expression in cultured human keratinocytes
AU Salo, Tuula; Lyons, J. Guy; Rahemtulla, Firoz; Birkedal-Hansen, Henning; Larjava, Hannu
CS Dep. Oral Surg., Univ. Oulu, Oulu, SF-90220, Finland
SO Journal of Biological Chemistry (1991), 266(18), 11436-41
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English

L9 ANSWER 37 OF 37 BIOTECHNO COPYRIGHT 2003 Elsevier Science B.V.
AN 1993:23273871 BIOTECHNO
TI Interleukin-1.beta. and transforming growth factor-.alpha./epidermal growth factor induce expression of M(r) 95,000 type IV collagenase/gelatinase and interstitial fibroblast-type collagenase by rat mucosal keratinocytes

AU Lyons J.G.; Birkedal-Hansen B.; Pierson M.C.; Whitelock J.M.; Birkedal-Hansen H.
CS Dept. of Oral Biology, Univ. of Alabama School of Dentistry, SDB Box 54,Birmingham, AL 35294, United States.
SO Journal of Biological Chemistry, (1993), 268/25 (19143-19151) CODEN: JBCHA3 ISSN: 0021-9258
DT Journal; Article
CY United States
LA English
SL English

L9 ANSWER 30 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 94:381608 SCISEARCH
GA The Genuine Article (R) Number: NR016
TI INDUCTION OF MEMBRANE RUFFLING BY GROWTH-FACTORS IN MORPHOLOGICALLY TPA-RESISTANT BALB/C3T3 TR4 CELLS
AU ENOMOTO T (Reprint); ASANO Y
CS KOBE UNIV, SCH MED, DEPT RADIAT BIOPHYS & GENET, CHUO KU, KUSUNOKI 7-5-1, KOBE 650, JAPAN (Reprint); OSAKA UNIV, MICROBIAL DIS RES INST, DEPT ONCOGENE RES, SUITA, OSAKA 565, JAPAN
CYA JAPAN
SO CELL STRUCTURE AND FUNCTION, (APR 1994) Vol. 19, No. 2, pp. 89-96. ISSN: 0386-7196.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 32
*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

L9 ANSWER 23 OF 37 MEDLINE DUPLICATE 10
AN 96130535 MEDLINE
DN 96130535 PubMed ID: 8544402
TI Induction of plasminogen activator inhibitor type 1 in murine lupus-like glomerulonephritis.
AU Moll S; Menoud P A; Fulpius T; Pastore Y; Takahashi S; Fossati L; Vassalli J D; Sappino A P; Schifferli J A; Izui S
CS Department of Pathology, University of Geneva Medical School, Switzerland.
SO KIDNEY INTERNATIONAL, (1995 Nov) 48 (5) 1459-68. Journal code: 0323470. ISSN: 0085-2538.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199602
ED Entered STN: 19960227
Last Updated on STN: 19960227
Entered Medline: 19960214

L9 ANSWER 20 OF 37 MEDLINE DUPLICATE 8
AN 97081970 MEDLINE
DN 97081970 PubMed ID: 8923213
TI Increased expression of extracellular matrix proteins and decreased expression of matrix proteases after serial passage of glomerular mesangial cells.
AU Schnaper H W; Kopp J B; Poncelet A C; Hubchak S C; Stetler-Stevenson W G; Klotman P E; Kleinman H K
CS Department of Pediatrics, Northwestern University Medical School, Chicago, IL 60611-3008, USA.
NC R01-DK49362 (NIDDK)
SO JOURNAL OF CELL SCIENCE, (1996 Oct) 109 ( Pt 10) 2521-8. Journal code: 0052457. ISSN: 0021-9533.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Priority Journals
EM 199705
ED Entered STN: 19970523
Last Updated on STN: 19980206
Entered Medline: 19970515

L9 ANSWER 4 OF 37 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 2002:320800 BIOSIS
DN PREV200200320800
TI Bradykinin reduces renal interstitial fibrosis by increasing extracellular matrix degradation.
AU Schanstra, Joost P. (1); Drogoz, Pascale (1); Desmond, Laurence (1); Calise, Denis (1); Neau, Eric (1); Girolami, Jean-Pierre (1); Bascands, Jean-Loup (1)
CS (1) U388, INSERM, Toulouse Cedex 4 France
SO Journal of the American Society of Nephrology, (September, 2001) Vol. 12, No. Program and Abstract Issue, pp. 716A. http://www.jasn.org/. print.
Meeting Info.: ASN (American Society of Nephrology)/ISN (International Society of Nephrology) World Congress of Nephrology San Francisco, CA, USA October 10-17, 2001
ISSN: 1046-6673.
DT Conference
LA English

L9 ANSWER 2 OF 37 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 2002:390963 SCISEARCH
GA The Genuine Article (R) Number: 538VD
TI Expression of glomerular plasminogen activator inhibitor type 1 in glomerulonephritis
AU Hamano K; Iwano M (Reprint); Akai Y; Sato H; Kubo A; Nishitani Y; Uyama H; Yoshida Y; Miyazaki M; Shiiki H; Kohno S; Dohi K
CS Nara Med Univ, Dept Internal Med 1, 840 Shijo, Kashihara, Nara 6348522, Japan (Reprint); Nara Med Univ, Dept Internal Med 1, Kashihara, Nara 6348522, Japan; Nagasaki Univ, Sch Med, Dept Internal Med 2, Nagasaki 852, Japan
CYA Japan
SO AMERICAN JOURNAL OF KIDNEY DISEASES, (APR 2002) Vol. 39, No. 4, pp. 695-705.
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399 USA.
ISSN: 0272-6386.
DT Article; Journal
LA English
REC Reference Count: 55
*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

L10 ANSWER 1 OF 5 MEDLINE
AN 1999062262 MEDLINE
DN 99062262 PubMed ID: 9844133
TI Targeting TGF-beta overexpression in renal disease: maximizing the antifibrotic action of angiotensin II blockade.
AU Peters H; Border W A; Noble N A
CS Division of Nephrology, University of Utah School of Medicine, Salt Lake City, Utah, USA.
NC DK 43609 (NIDDK)
DK 49342 (NIDDK)
DK 49374 (NIDDK)
SO KIDNEY INTERNATIONAL, (1998 Nov) 54 (5) 1570-80.
Journal code: 0323470. ISSN: 0085-2538.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals

EM 199902
ED Entered STN: 19990223
Last Updated on STN: 19990223
Entered Medline: 19990211